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ABSTRACT: We analyzed the effects of gravitational unloading on muscular fatigability and the effectiveness of resistive vibration exercise to counteract these changes. Changes in knee extensor fatigability as a consequence of 8 weeks of horizontal bedrest with or without daily resistive vibration exercise were evaluated in 17 healthy male volunteers. Bedrest increased fatigability (% decrease in maximal voluntary isometric torque per minute exercise) from –7.2 ± 0.5 to –10.2 ± 1.0%/min ($P < 0.05$), which was accompanied by a decline (of 52.0 ± 3.7%, $P < 0.05$) in muscle blood flow. Daily resistive vibration exercise training during bedrest prevented increases in fatigability (from –10.8 ± 1.8 to –8.4 ± 1.6%/min, $P < 0.05$), and mitigated the reduction in blood flow (decline of 26.1 ± 5.1%, $P < 0.05$). Daily resistive exercise may thus be suggested as an effective countermeasure during spaceflight and illness-related prolonged bedrest to combat the detrimental changes in muscle endurance that result from gravitational unloading.

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KNEE EXTENSOR FATIGABILITY AFTER BEDREST FOR 8 WEEKS WITH AND WITHOUT COUNTERMEASURE

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The adaptation of skeletal muscle due to gravitational unloading extends beyond a mere downsizing of the contractile apparatus, and includes shifts in myosin phenotype[1,35] as well as cardiovascular adaptations.[8] Hence, reduced oxygen delivery and oxygen utilization may impair the capacity for prolonged exercise following unloading. Exercise tolerance may be further influenced by impaired muscle activation after gravitational unloading.[13,17] It therefore seems reasonable to expect a deterioration in exercise tolerance as a consequence of simulated or actual spaceflight. However, some investigators have observed increased local muscle fatigability following gravitational unloading,[14,25,26] whereas others have reported unchanged[6,36] or even reduced[10,31] muscle fatigability. These inconsistencies might be partly related to methodological differences between studies and between individual fatigue protocols. Information about possible underlying mechanisms is important for understanding bedrest-induced changes in fatigability, which may help to develop effective preventative measures.

The primary purpose of the present study was to test the hypothesis that the fatigability of the human quadriceps femoris muscle is significantly impaired after prolonged horizontal bedrest. This hypothesis was evaluated by means of a 5-min intermittent sub-

Abbreviations: BFI, Blood flow index; Fmed, median frequency; MFCV, muscle-fiber conduction velocity; MVT, maximal voluntary torque; RMS, root mean square; RVE, resistance vibration exercise group; sEMG, surface electromyography; TOI, tissue oxygenation index; TTI, time–torque integral

Key words: blood flow; high-density surface EMG; intermittent exercise; near-infrared spectroscopy; surface EMG; unloading

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maximal isometric knee extension fatigue protocol. To locate causes of altered fatigability both at the central and peripheral level, we recorded surface electromyographic (sEMG) signals from the lateral vastus muscle during the fatiguing exercise. In addition, near-infrared spectroscopy was used to determine whether bedrest-induced changes in fatigability were related to changes in local muscle oxygenation and blood flow.

The major preventative measure to offset the deconditioning effects of microgravity is physical exercise.[7] Resistive vibration exercise, which comprises both resistance exercise and vibration exercise, has recently been proposed as a promising training modality to preserve bone mass and maintain muscle mass, strength, and endurance capacity.[27] The second major objective of the present study, therefore, was to determine whether changes in fatigability of the knee extensor could be counteracted by daily resistive vibration exercise.

MATERIALS AND METHODS

Subjects. Twenty male volunteers participated in the study. At the start of the study the subjects were randomly assigned to a training group or an inactive control group. The training group [resistance vibration exercise (RVE), $n = 10$; mean age, height, and body mass ± SD: 32.7 ± 4.8 years, 186.3 ± 8.0 cm, and 86.5 ± 16.5 kg, respectively] participated in a progressive resistive vibration exercise program during the bedrest. The subjects of the control group ($n = 10$; mean age, height, and body mass ± SD: 33.4 ± 6.6 years, 185.4 ± 7.7 cm, and 79.7 ± 10.9 kg, respectively) were restricted to bedrest without countermeasure. Subjects did not participate in any specific training/exercise program prior to the start of the study and the average exercise activity (h/wk) prior to the start of the study was similar for the RVE (2.6 ± 2.4) and control group (2.4 ± 3.6). The study received approval of the local ethics committee and all participants gave written informed consent.

General Design. Subjects were confined to 56 days of strict horizontal bedrest at the Benjamin Franklin Hospital as previously described.[27] Ingestion of alcohol or nicotine, as well as the regular intake of medication, was prohibited.

Exercise-Based Countermeasures. RVE subjects performed resistive exercise on a vibration system that was specifically developed for application under microgravity and bedrest conditions (Galileo Space; Novotec, Pforzheim, Germany). The applied equipment and protocol for countermeasure exercise are described in detail elsewhere.[27] In short, the training device consists of a vibration platform, which is vertically suspended on a trolley. Elastic springs were attached to the trolley for the subjects to attach themselves through belts with their shoulders, hips, and hands. During bedrest, RVE subjects trained two times daily for 6 days per week. In each training session, four resistive exercises were performed in the following order: squats, heel raises, toe raises, and explosive squats. All exercises were performed while the platform was vibrated at a frequency of 19 Hz. Vibration frequency was progressively increased to ~26 Hz at the end of bedrest.[27]

Measurement Procedures. Subjects were tested on two occasions: baseline measurements were performed 1 day prior to the start of bedrest. The post-bedrest measurement was scheduled on the third day of reambulation. The design of the measurements, which were performed in supine position, has been previously described.[24] The hips were flexed to ~115°. The knee pits were supported by a padded rigid horizontal bar and the subject's left and right feet were strapped in custom-built padded cuffs, with the ankle joints in a neutral position. The cuff of the right leg was connected to a force transducer (KAP-E/2kN, A.S.T., Dresden, Germany) that was mounted on a rigid horizontal bar and oriented perpendicular to the line of pull of the lower leg. The distance between the transducer and the axis of the knee joint (moment arm) was adjusted for each subject and was kept constant between experiments. The knee flexion angle was set at the individually determined optimal angle (either 60° or 70°)[24] and remained constant between experiments. The pelvis and upper body were securely fixed to the dynamometer by belts. Isometric force was recorded during voluntary contractions of the quadriceps femoris muscle of the right leg. Force signals were digitized using a sampling rate of 1,000 samples/s and stored to disc for off-line analysis. Knee extension torque was calculated as the product of force and moment arm.

Subjects started each experimental session by performing a specific warm-up set as previously described.[24] Following the warm-up, the subjects were asked to maximally exert isometric torque for 2–4 s. Three to five maximal attempts were made, separated by a minimum of rest for 2 min. The highest force was taken to calculate the maximal voluntary torque (MVT).

Fatigue Task. We designed a 5-min intermittent endurance test that was anticipated to be difficult but not impossible to complete after 56 days of bedrest. An intermittent submaximal isometric protocol was considered more appropriate to detect changes in skeletal muscle fatigability than a sustained test protocol, because intermittent exercise (1) is metabolically more demanding than continuous exercise,[29] and (2) relies more on aerobic energy supply as compared to sustained exercise, which partly or completely precludes blood flow.[32] Based on pilot studies, the target torque during the main measurements was set at 45% of the actual MVT, i.e., at the same relative contraction intensity, pre- and post-bedrest. Setting the exercise intensity at a relative target torque level allowed the investigation of qualitative changes in fatigue characteristics, irrespective of changes in maximal torque-generating capacity. The subjects were expected to perform in total 120 submaximal contractions in five consecutive exercise blocks, each of 1 min duration. Each block consisted of 24 repeated isometric contractions that were sustained for 1.5 s with 1 s of rest between them (Fig. 1A). During the submaximal contractions, both visual (target level) and auditory (timing) feedback was given. Within 2 s after each exercise block, subjects were instructed to perform a single MVC that was 2–4 s in duration. In cases where subjects, due to exhaustion, failed to reach the target torque during the submaximal contractions in three consecutive attempts, they finished the ongoing exercise block and performed a final MVC, after which the fatigue test was ended.

Surface EMG Acquisition. High-density surface EMG (HD-sEMG) signals were recorded from the distal one-third of the right vastus lateralis muscle by means of a high-density sEMG system (Active One; BioSemi, Amsterdam, The Netherlands). The system consisted of 130 densely spaced skin-surface electrodes (5-mm interelectrode distance), arranged in a rectangular 10 × 13 matrix. The columns of 13 electrodes were aligned parallel to the muscle-fiber orientation of the vastus lateralis with the motor endplate zone around the center of the columns.

Voluntary Torque and Surface EMG Data Processing. Voluntary torque and sEMG data obtained during submaximal contractions were processed only when the peak voluntary torque during these attempts exceeded 30% of the initial MVT. Unexpectedly, not all subjects were able to perform all 120 submaximal contractions before bedrest. In addition, the number of completed exercise blocks and performed

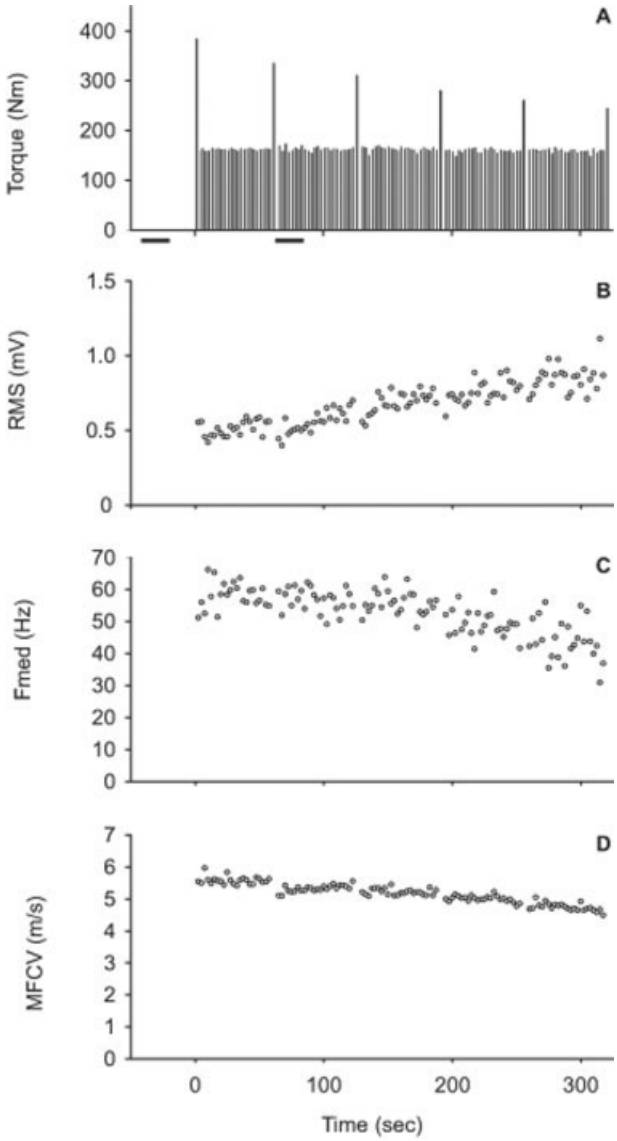

FIGURE 1. Representative torque and sEMG profiles of one subject during the fatigue task. The vertical bars in **(A)** represent the voluntary torque, the open circles in **(B–D)** represent the different sEMG values (respectively RMS, root means square; Fmed, median frequency; MFCV, muscle-fiber conduction velocity) obtained during the intermittent submaximal isometric contractions. Near-infrared spectroscopy recordings were obtained at rest and during the first 20 s of the second consecutive exercise block (horizontal bars in **A**).

submaximal contractions varied between sessions (see Table 1 for group values). Hence, a complete dataset consisting of 120 submaximal and 6 maximal voluntary contractions, for both pre- and post-bedrest, was obtained only for five control and five RVE subjects. Torque and sEMG data were analyzed by linear regression as a percentage rate of change of initial value per minute of exercise.[26] In incomplete tests due to early exhaustion, regression of MVT data

Table 1. Mean values of changes in time–torque integral and the number of exercise blocks completed, pre- and post-bedrest.

	Controls	RVE	Significant group by time interaction
Change in TTI, %/min			
Pre-BR	–4.4 ± 1.4	–6.1 ± 2.2	
Post-BR	–6.0 ± 1.3	–3.3 ± 1.8*	†
Completed exercise blocks			
Pre-BR	4.9 ± 0.1	4.3 ± 0.3	
Post-BR	4.6 ± 0.2	4.8 ± 0.1	†

RVE, resistance vibration exercise; TTI, time–torque integral; BR, bedrest. Values are mean ± SEM.
**Significant difference between pre- and post-bedrest (BR).*
†Significant time course difference between controls and RVE group ($P < 0.05$).

was derived from 4–6 data points, whereas regression of torque and sEMG variables was derived from 72–120 data points.

The fatigability of the quadriceps muscle was defined as the percentage decline in MVT from initial value per minute exercise. To check for differences in task performance, the normalized time–torque integral (TTI) was calculated for each submaximal contraction, which was defined as the area under the normalized torque–time curve.[29] Systemic changes were sought by regression analysis. The RMS was calculated over a 1-s interval for each maximal voluntary contraction. sEMG signals were also analyzed for submaximal contractions when voluntary torque exceeded 30% of the baseline MVT. For each submaximal segment the sEMG was quantified for signal amplitude (RMS) and median frequency (Fmed) from a monopolar recording. Muscle-fiber conduction velocity (MFCV) estimates were derived from bipolar recordings and calculated from the time delay between two differential signals, spaced in a double interelectrode distance[2] of 10 mm. MFCV values were calculated only when correlation coefficients between consecutive bipolar signals exceeded 0.8 and the MFCV values obtained were less than 8.0 m/s. Reliable MFCV estimates were obtained for seven control subjects and seven RVE subjects. All sEMG variables were averaged for each column; the column with the highest mean RMS was selected and the mean sEMG values of this column were used in the regressions.

Near-Infrared Spectroscopy. Local oxygenation and hemodynamics of the vastus lateralis were monitored by near-infrared spectroscopy, using a triple-wavelength continuous wave spectrophotometer (NIRO 300; Hamamatsu Photonics, Hamamatsu City, Japan) with an emitting optode and a corresponding receiver optode at an interoptode distance of 5.0 cm. The optode was placed directly proximal to the sEMG grid over the right vastus lateralis muscle.

In the NIRO 300, three pulsed laser diodes provide light in the near-infrared range at wavelengths $\lambda = 778$, 813, and 853 nm. Attenuation of scattered light was detected for each near-infrared wavelength at a sampling rate of six samples/s. Hemoglobin oxygenation in the vastus lateralis was measured by spatially resolved reflectance spectroscopy[34] as tissue oxygenation index (TOI) reflecting the mean hemoglobin oxygen saturation in the scanned tissue section.[4] Local muscle blood flow was estimated from the kinetics of an intravenous bolus of indocyanine green (PulsionMedical Systems, Munich, Germany) from which a relative blood flow index (BFI) was calculated.[19] TOI was assessed prior to the administration of the tracer. TOI was thereby calculated as the mean value over 5 s.

Hemoglobin oxygenation and blood flow were assessed at rest and during the first 20 s of the second consecutive block (see Fig. 1A) of the fatigue exercise protocol.

Statistical Analysis. Data are presented as means ± SEM. Differences in the response to bedrest between the RVE and the control groups were tested with repeated measures ANOVA (SPSS 12.0; Chicago, Illinois). If a time-by-group interaction was detected, paired-sample *t*-tests between pre- and post-bedrest data were performed within groups, and unpaired *t*-tests were performed to test for differences between groups. Pearson's correlation coefficients were calculated to test for correlations. Differences were considered statistically significant at $P < 0.05$.

RESULTS

In two subjects of the control group, sEMG recordings were not obtained during the post-bedrest session either due to improper fixation of the sEMG grid or due to muscle cramps. A third subject (RVE group) could not participate in either session because of patellar discomfort during maximal isometric contractions. Data of these subjects were discarded from the analyses. Results are thus presented for 17 subjects (8 control subjects and 9 RVE subjects), except for MFCV estimates, which were based on 14 subjects (7 control, 7 RVE).

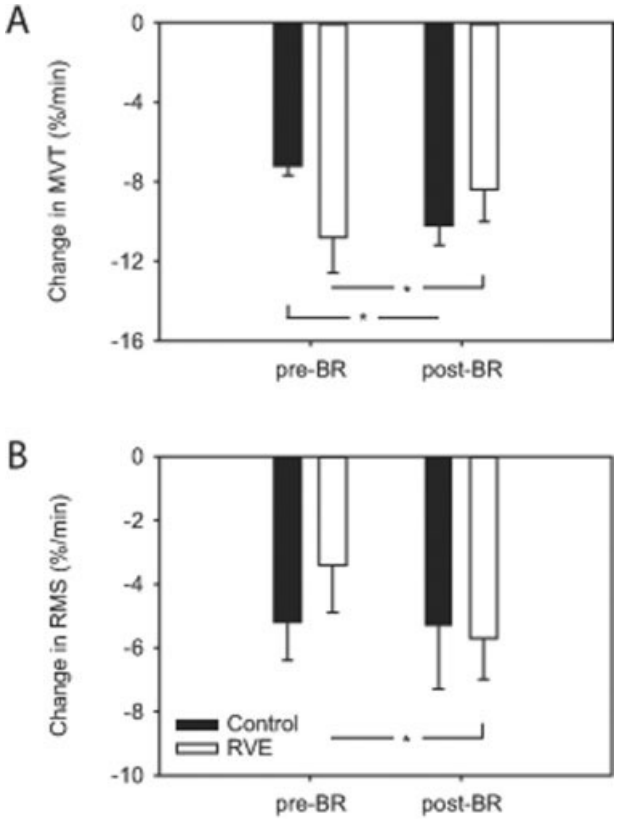

FIGURE 2. Fatigability **(A)** of the control and RVE group expressed as the percent change of initial maximal voluntary torque (MVT) per minute exercise and **(B)** the percent change per minute exercise from the initial maximal root mean square (RMS) of the sEMG obtained during the maximal contractions, before and after bedrest (BR). Values are expressed as mean ± SEM. *Significant difference ($P < 0.05$) between pre- and post-bedrest.

Voluntary Torque. The initial (nonfatigued) MVT decreased as a consequence of bedrest for both groups (both $P < 0.01$). For controls it decreased from 317 ± 17 to 267 ± 18 Nm, and for the RVE group from 300 ± 14 to 268 ± 17 Nm. The change in MVT between the days of testing was not significantly different between groups. Fatigability was enhanced following bedrest without countermeasures. RVE training not only prevented this effect, but even induced a reduction in fatigability ($P < 0.05$, Fig. 2A). TTI declined during the repeated contractions in all conditions (Table 1). For controls the TTI rate of decline was similar during pre- and post-bedrest conditions, whereas for RVE a slightly smaller decline was observed post-bedrest. The groups also responded oppositely ($P < 0.05$) with respect to the number of blocks that the subjects performed prior to and after bedrest. Two control subjects performed one block less following bedrest, as compared to before, whereas three RVE subjects performed one or two blocks more following bedrest. The changes within the groups did not reach statistical significance at the group level (Table 1).

Surface Electromyography. Representative changes in sEMG variables during the fatigue protocol are given in Figure 1B–D.

The sEMG amplitude (RMS) decreased during maximal voluntary contractions (Fig. 2B, all $P < 0.05$) and increased during submaximal contractions for each fatigue protocol in both groups (Fig. 3A, all $P < 0.05$). The rate of decrease in maximal RMS was significantly greater in RVE after than before bedrest ($P < 0.05$), whereas no changes were seen for controls. The rate of increase in submaximal RMS was similar among groups during both sessions and no changes were observed as a consequence of bedrest. The mean RMS determined over the last 10 submaximal contractions did not differ significantly between controls and RVE prior to (66.7 ± 5.7 vs. 63.4 ± 5.3%) or after bedrest (76.1 ± 5.6 vs. 74.7 ± 7.5%). In addition, no changes were observed between sessions.

The median frequency (Fmed) decreased during submaximal contractions in both groups, both before and after bedrest (Fig. 3B, all $P < 0.01$). In the absence of countermeasures, the decline in median frequency was greater after bedrest than pre-bedrest ($P < 0.05$). RVE training prevented this difference.

For controls, MFCV decreased as a consequence of the repeated submaximal contractions, both before and after bedrest ($P < 0.05$), and the rate of decline was enhanced ($P < 0.05$) post-bedrest (Fig. 3C). In contrast, for RVE the MFCV decline was not significantly affected by the test protocol, either before or after bedrest.

Near-Infrared Spectroscopy. At rest, BFI and TOI did not differ between groups prior to the start of bedrest. BFI was lower ($P < 0.05$) after bedrest in both groups, whereas no significant change was observed in muscle TOI at rest (Table 2).

Compared to resting conditions, BFI was elevated during exercise, whereas TOI was reduced (Table 2). In controls, BFI and TOI were markedly lower after than before bedrest ($P < 0.05$ each). BFI was also reduced after bedrest for RVE ($P < 0.05$), yet the reduction was attenuated when compared to the control group ($P < 0.01$). No changes were seen in TOI during exercise for RVE. As such, post-bedrest values for BFI and TOI were higher for RVE than those obtained for control subjects ($P < 0.001$ each). As shown in Figure 4, significant negative correlations were found between the relative pre- to post-bedrest change (i.e., the percent change from baseline) in BFI

and fatigability ($P < 0.01$, r = 0.68, $n = 17$, Fig. 4A) as well as between the relative changes in TOI and fatigability ($P < 0.01$, $r = 0.65$, $n = 17$, Fig. 4B).

DISCUSSION

Bedrest-Induced Changes in Fatigability. We assessed the effect of bedrest for 8 weeks on the resistance to fatigue of the quadriceps femoris muscle. The fatigability of the quadriceps was significantly accelerated by ~50% after the bedrest period. The ability to maintain torque output during repeated voluntary contractions depends on intrinsic/metabolic properties of the muscle fibers, local blood flow providing the muscle with oxygen, as well as neural activation.

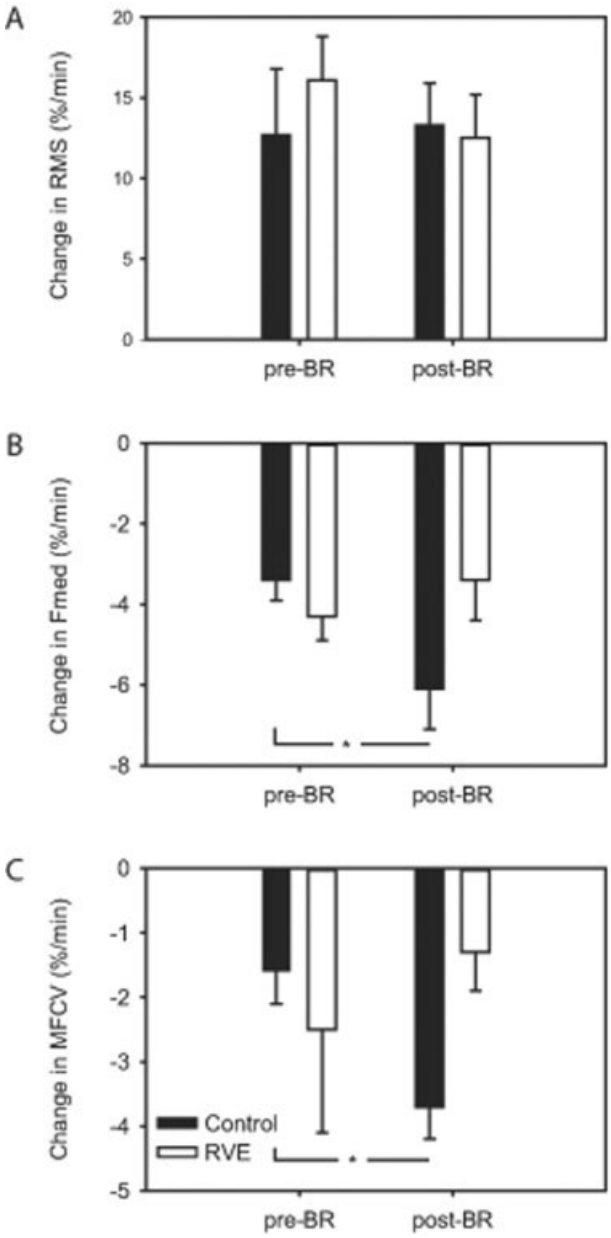

FIGURE 3. Changes in sEMG amplitude **(A)**, median frequency (Fmed; **B**) and muscle-fiber conduction velocity (MFCV; **C**) for the control and RVE groups as a consequence of the fatiguing submaximal contractions, pre- and post-bedrest (BR). Values (mean ± SEM) are expressed in percent change of initial value per minute exercise. *Significant difference ($P < 0.05$) between pre- and post-bedrest (BR).

Table 2. Blood flow and tissue oxygenation measured by near-infrared spectroscopy during rest and exercise, pre- and post-bedrest.

	Controls	RVE
BFI (nmol/L/s)		
Pre-BR		
Rest	8.2 ± 0.8	8.4 ± 0.9
Exercise	23.4 ± 0.7	23.3 ± 1.7
Post-BR		
Rest	5.6 ± 0.7*	7.3 ± 0.6*
Exercise	11.4 ± 0.9*†	17.0 ± 1.3*
TOI (%)		
Pre-BR		
Rest	73.7 ± 0.6	73.3 ± 0.5
Exercise	54.1 ± 1.2	53.2 ± 1.3
Post-BR		
Rest	73.4 ± 1.0	72.2 ± 2.1
Exercise	42.1 ± 1.2*†	54.9 ± 1.7

RVE, resistance vibration exercise; BFI, blood flow index; TOI, tissue oxygenation index.
Values are means ± SEM.
**Significantly lower post-bedrest (BR) compared to pre-BR values (P < 0.05).*
†Significantly lower as compared to RVE (P < 0.05). Compared to resting conditions, BFI was significantly elevated during exercise, whereas TOI was significantly reduced in both groups.

sEMG Profiles during the Fatigue Task. RMS of the EMG signals declined during maximal voluntary contractions. This finding is consistent with the loss in sEMG signal amplitude during sustained maximal contractions,[28] and can be explained by the development of central activation failure,[30] the decrease in MFCV,[33] or a reduced mean motor unit firing frequency.[22] In contrast, central drive was intensified during the submaximal contractions, as indicated by the increase in RMS during these contractions (Fig. 3A). The increase in submaximal sEMG amplitude reflects compensation for the fatigue-induced loss in force output of individual motor units by the recruitment of additional motor units and modulation of the discharge rate of the motor units.[15,23,28] In addition, a fatigue-induced increase in synchronization of motor unit firing patterns[18] may contribute to the increase in RMS. No consistent changes were observed in maximal or submaximal RMS profile, suggesting that a similar percentage of the maximal

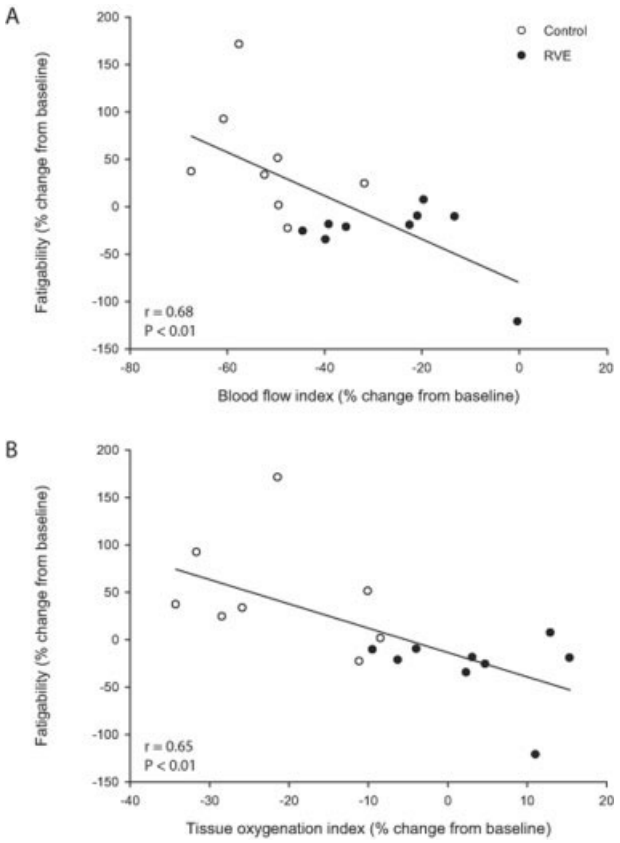

FIGURE 4. Correlation between bedrest-induced changes in muscle blood flow and fatigability **(A)** and correlation between bedrest-induced changes in tissue oxygenation index and fatigability **(B)**. Individual values (n = 17) are expressed as percent changes from baseline. Both linear regressions were significant.

instantaneous neural capacity was present toward the end of each fatigue protocol. Indeed, the mean RMS of the 10 last submaximal contractions was not significantly different before and after bedrest (66.7 ± 5.7% vs. 76.1 ± 5.6%). This suggests that changes in neural activation[31] cannot explain the increased fatigability in the control group after bedrest.

Two additional sEMG variables frequently used to rate local muscle fatigue are the median frequency (Fmed) of the power density spectrum and the mean MFCV. Consistent with previous reports,[23] MFCV decreased during the pre- and post-bedrest fatigue protocol (Fig. 4B,C), which reflects the deteriorating metabolic status of the muscle.[21] The finding that both Fmed and MFCV declined at a faster rate following bedrest suggests that an accelerated development of peripheral fatigue occurred following bedrest.

Muscle Blood Flow and Oxygenation. The muscle TOI reflects the local balance between oxygen delivery and consumption.[5] A decrease in regional blood flow may underlie an increased peripheral fatigability if TOI is similarly reduced, indicating an increased local oxygen extraction.

The present data confirm that muscle blood flow increases during repeated isometric contractions (Table 2).[20] More important, muscle blood flow during exercise was reduced by ~50% following bedrest (Table 2), suggesting a severe restriction in oxygen delivery. The diminished perfusion during exercise cannot be related solely to the reduction in target torque, since changes in blood flow markedly exceeded the reduction in target torque, and since the effects of bedrest on muscle blood flow were attenuated in the RVE group. The limitation in oxygen supply was in part compensated for by a higher relative extraction of oxygen, as reflected by a diminished TOI.[12] Negative correlations between bedrest-induced changes in fatigability and changes in blood flow and tissue oxygenation suggest that the greater rate of peripheral fatigue following bedrest reflects an increased need for anaerobic energy supply. This notion is supported by the data of Grichko et al.,[14] who demonstrated increased glycolytic activity during exercise, following gravitational unloading in rats.

In addition to its effect on oxygen delivery, the reduction in blood flow may have also reduced the capacity to washout metabolic waste products following bedrest. As oxidative capacity is diminished in acidic environments,[16] the lack of sufficient blood flow may thus have caused a faster fatigue following bedrest, because it limited oxidative metabolism both directly and indirectly.

Confounding Factors in Experimental Models of Muscle Fatigability. The observed deteriorated fatigue resistance is in line with other reports of increased skeletal muscle fatigability following different models of gravitational unloading, including hindlimb unloading in rats,[14] bedrest,[26] and space flight[25] in humans. Yet muscle fatigability has also been reported to remain unchanged[11,36] or even to decrease[10,31] following gravitational unloading in both humans and rats. These inconsistencies may in part be attributable to differences in the models and durations of unloading, and in gender, species, and muscles tested. In addition, the methodology used to induce muscle fatigue may also explain differences. In contrast to intermittent contractions, sustained contractions may lead to partial or complete occlusion of blood vessels[38] and thus exclude the potential effects of structural and functional (cardio)vascular changes.[9,37] In addition, fatigability might be overes-

timated when the target torque is fixed between experiments,[26] whereas normalizing the target torque to the actual maximal voluntary torque may result in underestimation of relative fatigability[10,11] if neural activation following unloading is reduced. Such a bias can be excluded in the present study, because neural deconditioning was not observed during bedrest for either group.[24]

Efficacy of Resistive Vibration Exercise. Exercise training is used as the primary preventive measure to preserve human physiological systems that are otherwise deconditioned by space flight.[7] We demonstrate that RVE not only effectively counteracted the reduction of exercise tolerance, but significantly reduced muscle fatigability following bedrest, as indicated by an attenuated loss of maximal knee extension torque (Fig. 2A). These changes occurred despite a lessened reduction in TTI during exercise after bedrest. This would suggest that the RVE subjects performed slightly better in terms of relative workload, and hence that their enhanced fatigue resistance following bedrest is in fact underestimated. The increased exercise tolerance in RVE subjects at the peripheral level was further reflected in unaltered Fmed and MFCV profiles during the submaximal contractions (Fig. 3). A paradoxical finding in the present study was the faster rate of decline in RMS obtained at the maximal attempts post-bedrest (Fig. 2B). The concept of muscle wisdom states that a decline in alpha motor neuron discharge rate during fatiguing contractions serves to maintain force by protecting against conduction failure and by optimizing the input to motor units as their contractile properties change.[22] Such a decline in discharge rate could explain why the RMS of the maximal attempts declined faster during the post-bedrest fatigue task without additional loss in maximal voluntary torque. Yet in both the RVE and the control groups, the mean RMS of the 10 last submaximal contractions was similar between before and after bedrest (63.4 ± 5.3% vs. 74.7 ± 7.5%). This indicates that both groups exercised at an equal percentage of their maximal neural capacity at the end of each task.

RVE training limited the reduction in muscle blood flow and increased oxygenation during exercise compared to control subjects (Fig. 4). Although RVE did not fully preserve muscle blood flow at exercise, the absence of changes in TOI suggests that tissue oxygenation and thus oxidative metabolism during exercise were not critically limited after bedrest in the RVE subjects. The previous finding that the current exercise regime mitigated the reduction in arterial femoral diameter in the trained group[3] further suggests sufficient tissue perfusion maintenance in the RVE trained subjects. Thus, RVE may have preserved muscle endurance in part due to attenuation of structural and functional changes in the muscle (micro)vasculature.[3]

In conclusion, the present study demonstrates that the fatigability of the quadriceps femoris muscle during voluntary intermittent submaximal isometric knee extension was significantly increased following 8 weeks of bedrest. In combination, sEMG and near-infrared spectroscopy data suggest that the enhanced fatigability following bedrest is primarily related to impaired blood flow resulting in an impaired oxidative capacity. The RVE countermeasure reduced fatigability, prevented changes in fatigue-related sEMG variables, and mitigated the changes in blood flow. Such a time-efficient exercise may therefore be an effective countermeasure to combat detrimental changes as a result of gravitational unloading during spaceflight or illness-related bedrest.

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